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LATE SENIOR ASSISTANT SURGEON TO THE OPHTHALMIC CLINIQUE OF PROF. VON JAEGER, IN  
VIENNA, AUSTRIA

Translated by Dr. A. SCHAPRINGER, New York



[Reprinted from the ARCHIVES OF OPHTHALMOLOGY, Vol. xiii., Nos. 3 and 4,  
1884.]

presented by the author





# A CASE OF SUDDEN AMAUROSIS FOLLOWED BY HOMONYMOUS SUPERIOR HEMIANOPIA.

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THE literature of altitudinal—*i. e.*, superior or inferior—hemianopia is very meagre. The writings of Mackenzie and Von Graefe contain only cursory allusions to this subject. The first case published in full was that of Mauthner (1872):

There was loss of consciousness and headache for three days, followed by a defect in the upper part of the field of vision in both eyes. This defect increased in the course of two years, and at the end of this time involved almost the entire upper outer quadrant of each side. The defects were bounded by a zigzag line running above the horizontal meridian but quite near to it. The defect was somewhat larger on the right side than on the left, the vertical boundary line of the former nearly reaching the vertical meridian. In the course of time vision was gradually restored, but the patient became subject to epileptic attacks and died suddenly.

A similar case of homonymous superior hemianopia was observed by Schoen. In 1873, Russell published a case of monocular superior hemianopia of the right eye followed by complete blindness of both; and, in 1876, Schweigger, one of homonymous superior hemianopia with diminished perception of light in the upper part of the field of vision.

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In all of these cases no changes could be seen with the aid of the ophthalmoscope. Other cases have been published, such as two by Knapp, and one each by Schoen and by Schweigger, in which the retina or the optic nerve was diseased, and which, therefore, were not properly cases of hemianopia.

This short list includes all the cases of altitudinal hemianopia which have hitherto been observed clinically. *Post-mortem* examinations were made in two instances only, viz.: in the case of Mauthner, where the result was negative, and in the one of Russell, who found a tumor at the base of the brain.

The following is a sketch of a case of homonymous superior hemianopia which I have observed during life. The description of the lesions found after death has been transcribed from the record-book of autopsies, and will be found very defective and unsatisfactory. That it is so, is due to the fact that the autopsy was performed without a knowledge of the peculiar and interesting clinical features of the case.

A laborer, fifty-four years old, was admitted to the ophthalmic wards of Professor Von Jaeger on June 27, 1881, on account of sudden loss of vision which had occurred on the previous day. His eyesight had never been very good, but his general health had always been excellent up to two years ago when he had an attack of acute articular rheumatism which was followed by shortness of breath. He denied ever having had syphilis, nor could any traces of it be found upon examination.

In the early part of the month of February, 1881, he accidentally fell from a ladder the distance of about ten feet, striking the ground with his occiput. He became unconscious, and remained so for several hours, bleeding from the mouth, nose, and ears. On coming to he complained of violent headache; there was neither vomiting nor paralysis. His recovery was both protracted and incomplete, for since that time he has felt a constant "pressure upon his brain," his memory and will-power have become impaired, and he has suffered from frequent attacks of headache. About Easter-time he had a stroke of apoplexy with left-sided hemiplegia, but was able to work again by the end of a few weeks.



On the day preceding his admission he was busy raking hay, when his eyesight suddenly became impaired. It grew worse for the next hour and a half, at the end of which time it was totally abolished.

*Status præsens*.—Patient tall, well nourished, of a large frame. The skull was markedly dolichocephalous. Head erect. Irides blue. Pupils equally and widely dilated, not responding to light. Eyeballs immovable, visual lines nearly parallel and slightly elevated. Media clear. Lids widely separated. Ophthalmoscope shows compound hyperopic astigmatism in both eyes, requiring for its correction about  $+ \frac{1}{12}$  in the vertical, and  $+ \frac{1}{8}$  in the horizontal meridian. Fundus otherwise normal—*i. e.*, no disease of either the retina or the optic nerve. Paresis of the left facial nerve, involving the twigs supplying the lips, nostrils, and eyelids. Deviation of tongue to the right. The sense of taste impaired upon the left side of the tongue. Sense of smell, as tested with spirits of turpentine, wanting. He answers questions correctly; his hearing power appears to be normal. The sensibility of the skin is nowhere diminished. The pressure of his hands is strong and equal on both sides. There is no unsteadiness in walking. Area of dulness over heart increased, heart-sounds louder than normal. Pulse 60, strong, jerking, and incompressible. Temperature normal. Intense headache.

Ordered: ice-cap to head, and iodide of potassium internally.

*June 28th*.—Headache unabated. Had been vomiting several times during the night.

*June 29th*.—Headache somewhat less. Patient says that he had been seeing a mist before his eyes since last evening. In the course of the afternoon he said that he could discern objects. A slight difference could now be noticed in the size of the pupils, the left not being quite so much dilated as the right. The left pupil reacted whenever light was thrown either into the left eye, or into the right, whilst the right pupil continued immovable. He counted fingers at fifteen feet with the left eye, and at twelve feet with the right.

*July 1st*.—Pupils equally and moderately dilated, each reacting well upon direct stimulus as well as in sympathy with the other. Headache gone.

His vision was again tested a few days afterward when he was found able to count fingers at the distance of eighteen feet with either eye. His color-perception was unimpaired. The upper

half of the visual field of both eyes was wanting. Subsequent examinations made with the aid of a perimeter showed that the boundary line of the defect in the right eye coincided with the horizontal meridian, whilst in the left eye it ran at a distance of eight or twelve degrees above it. In the latter eye a narrow zone of amblyopia appeared to exist between the lower preserved portion of the visual field and the upper portion in which vision was entirely abolished. In this zone a lighted candle could be perceived by the patient, but not a piece of white paper.

Binocular vision was preserved, as proved by the fact that he could always tell whether the convex or the concave side of a pair of curved scissors was presented to him. He never saw double. He could freely move both eyes downward as well as to the right or left, but not upward.

Subsequent repeated examinations elicited no change from the condition just described. After staying at the clinique for three months, he was transferred to the poorhouse.

The *diagnosis* made was: *Hemorrhage at the base of the brain in the neighborhood of the optic chiasm.* This diagnosis was based on the sudden onset of the amaurosis and the complete immobility of the pupils. These two symptoms might also have been produced by bilateral lesions of the optic tracts, the optic thalami, the geniculate bodies, the corona radiata, or the occipital lobes, but such locations were highly improbable, since one could not reasonably suppose that two distinct lesions, one in each hemisphere, should have occurred exactly at one and the same time. The corpora quadrigemina had to be excluded since there were no disturbances of coördination such as are said to occur invariably with lesions of this organ. The possibility of embolism instead of hemorrhage had also to be taken into consideration, but there were no circumstances in its favor, whilst the occurrence of hemorrhage was made plausible by the atheromatous condition of the arteries. Besides this, the traumatism occasioned by the fall from the ladder was also likely to have produced changes within the skull cavity predisposing to rupture of the vessels.

The theory of hemorrhage in the region of the optic chiasm was corroborated by the development of superior hemianopia, since the latter could easily be explained by the



pressure of the clot upon the fibres contained in the inferior portion of the optic nerves and corresponding to the upper part of the visual field. The phenomena exhibited by the pupils were also in harmony with our hypothesis. The paralysis of the facial nerve was considered as a remnant of the attack of hemiplegia which he had suffered before. Basilar hemorrhages are known to occasionally produce anosmia and ageusia. These two symptoms were present in our case and did not militate against our diagnosis.

The following notes of the remainder of the patient's history are the result of inquiries which I made among his relatives.

His general condition and that of his eyesight remained unchanged. He could go about alone and was able to visit his brother, who lived forty-five minutes' walk from the poorhouse. He had no further stroke of apoplexy. His death occurred nine months and a half after the sudden attack of blindness, and was caused by the incarceration of a hernia.

The result of the *post-mortem* examination performed by Dr. Weichselbaum, was recorded as follows :

Atheromatous changes of the arteries at the base of the brain. Old hemorrhagic foci in the temporal lobe, the lenticular nucleus, the medullary substance of the frontal lobe, and the cortical substance of the sulcus olfactorius of the right hemisphere, the parietal lobe and the optic thalamus of the left hemisphere. Chronic pachymeningitis.

Eccentric hypertrophy of the left ventricle of the heart.

Incarcerated scrotal hernia on the right side. Diphtheritic inflammation of the mucous and serous coats of the prolapsed portion of the intestine. Incipient inflammation of the remaining portions of the intestine.

Consolidation of the apex of the left lung. Firm pleuritic adhesions and emphysema of the right lung.

No mention is made in this record of hemorrhage in the neighborhood of the chiasm, the optic nerves or tracts or anywhere at the base of the brain, nor of fracture or fissure of the skull. Our diagnosis of hemorrhage in the neighborhood of the chiasm, therefore, is proved to have been

erroneous, but the lesions as described do not help to explain the symptoms observed during life.

The record furnishes no clue which would enable us to determine the chronological succession of the hemorrhages. The time which had elapsed after the hemorrhages had taken place had perhaps been too long, so that their relative dates could not be made out at the autopsy even if attention had been paid to this particular point. This is a serious drawback, and renders the difficult task of interpreting the clinical history still more difficult.

When the patient met with the accident by falling from a ladder, he became unconscious and bled from his mouth, nose, and ears. This was most probably due to a fracture of the base of the skull, and it is remarkable that no trace of it was found at the autopsy, but it is possible that it was overlooked. The pachymeningitis (which was recorded) could have been the consequence of such a fracture.

His memory and his will-power had been permanently impaired by the accident, but there was no paralysis at the time. We have to turn to the frontal lobes for an explanation of these clinical features.

Ferrier,<sup>1</sup> speaking of the effects of the removal or destruction by the cautery of the antero-frontal lobes in monkeys, says that these operations are not followed by any definite physiological results. "The sensory faculties, sight, hearing, touch, taste, and smell, remain unimpaired. The powers of voluntary motion are retained in their integrity, and there is little to indicate the presence of such an extensive lesion, or removal of so large a part of the brain." "And yet, notwithstanding this apparent absence of physiological symptoms, I could perceive a very decided alteration in the animal's character and behavior, though it is difficult to state in precise terms the nature of the change." "While not actually deprived of intelligence, they had lost, to all appearance, the faculty of attentive and intelligent observation." This faculty of attention Ferrier considers to be dependent upon the action of the inhibitory centres, since it is necessary to suppress every impulse of motion in

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<sup>1</sup> "The Functions of the Brain," p. 78.



order to concentrate attention. The development of the will-power is also dependent upon the development of the inhibitory centres, since with the progressive development of these centres the actions of the will lose impulsive character, and assume the appearance of conscious determination. In our case a clot was found in the right frontal lobe which was probably at the bottom of the loss of memory and will-power. It is reasonable to suppose that the hemorrhage was due to a lesion *by contrecoup* at the time when the patient fell from the ladder and struck the back of his head.

The hemorrhage found in the right lenticular nucleus was probably the cause of the left-sided hemiplegia which supervened a few months after the accident of the fall from the ladder. Hemiplegia is so frequently caused by hemorrhage into the lenticular nucleus, that in every case of hemiplegia we have first to think of hemorrhage in that region, and, as Nothnagel puts it, the odds are greater of finding it in that part of the brain than in any other part. Unless the clot is very small, hemiplegia invariably results. If the internal capsule is not involved, the hemiplegia soon disappears as it did in this case.

The next feature of the clinical history to be explained, in the light of the anatomical data, is the sudden amaurosis of both eyes, which supervened a short time after his recovery from the stroke of one-sided paralysis. No one of all the lesions found could have produced complete blindness singly, and the conclusion is forced upon us that it was the result of two simultaneous hemorrhages. The following combinations have to be taken into consideration:

1. *Right lenticular nucleus and left optic thalamus.*
2. *Right frontal lobe and left optic thalamus, and,*
3. *Right temporal lobe and left optic thalamus.*

Lesions of any of the single organs named may produce homonymous hemianopia, and the combinations enumerated would result in bilateral hemianopia of both eyes, *i. e.*, complete amaurosis.

The lesion of the right lenticular nucleus which enters into the first combination, having already been utilized to

explain the hemiplegia, ought not to be taken into consideration under this head. It is true that a lesion of the lenticular body can cause both hemianopia *and* hemiplegia, but in our case the hemiplegia preceded the blindness by several weeks.

*Second combination: right frontal lobe and left optic thalamus.* A clot in the frontal lobe, if sufficiently large, could compress the optic nerve of the same side and the chiasm, and by compression of one lateral half of the latter produce hemianopia. The lesion of the frontal lobe has, however, been made use of before as a substratum for the phenomena which were observed after the accidental fall from the ladder. Aside from this, there were other circumstances which render it improbable that the hemorrhage in this lobe should have had any share in the production of the visual disturbance. A clot formed in this locality would have exerted far more pressure upon the optic nerve itself than upon the chiasm. Consequently the right eye would have remained blind for some time after the inner half of the retina of the left eye had regained its power. This, however, was not the case. The right eye began to recover simultaneously with the left, and light thrown into the former elicited contraction of the left pupil. If the right optic nerve had been involved alone, the blindness would not have been complete, as the function of the nasal half of the left retina would not have been abrogated.

*Third combination: Right temporal lobe and left optic thalamus.* A hemorrhagic clot in the right temporal lobe, sufficiently large to exert pressure upon either the optic tract, the thalamus, the lenticular nucleus, or the corona radiata, would cause left-sided hemianopia. According to Ferrier the centres for smell and for taste are located at the apex of the temporal lobe. In our case both these senses were impaired. The fact that no motor disturbance was present does not militate against it, since it is an established fact that lesions of the temporal lobe, while producing in some instances the same motor symptoms as do lesions of the lenticular nucleus, will, in others, not produce any.

It is improbable that a clot capable of exerting so much



pressure should not also have compressed the right peduncle or the corpus striatum according to its situation. The implication of the former would in all probability have caused hemiplegia and that of the latter would certainly have done so. Hemiplegia, however, did not coexist with the blindness. Neither was there any hemianæsthesia of the face which is always associated with anosmia and ageusia whenever these defects are caused by lesions of the temporal lobe. It is possible, after all, that the clot in the temporal lobe caused only partial compression of the peduncle, in which case the amaurosis was caused by the clot in this lobe in conjunction with that found in the right thalamus.

An hypothesis which could explain the amaurosis on the basis of a single lesion would certainly be more plausible than any based on the assumption of simultaneous bilateral lesions. Let us see whether the focus found in the left optic thalamus could not in itself explain the ocular symptoms. The hemorrhage evidently did not take place in the posterior third of that organ, since a lesion in that place would have produced hemianopia of the opposite side, whilst we had to do with complete amaurosis. We must therefore locate the clot in the two anterior thirds of the thalamus. There a clot may exist without causing any disturbance of vision. But let us suppose that it was large enough to exert pressure upon the right as well as the left anterior tubercles of the corpora quadrigemina, but without influencing the posterior tubercles. The result would have been amaurosis, dilation with immobility of the pupils and immobility of the eyeballs—just the clinical picture observed in our case. The optic thalamus being a relatively small portion of the encephalon, a clot contained within its limits must necessarily also have been of limited dimensions, even though it may have compressed the anterior corpora quadrigemina, and capable of rapid absorption. This inference harmonizes with the prompt improvement which took place in the ocular symptoms. The conjugate upward paralysis, which remained long after the other muscles of the eyeballs had regained their power, also points to the im-

plication of the anterior tubercles of the corpora quadrigemina.

The objection might be raised against this explanation, that since according to it the left anterior tubercle was more involved than the right, right-sided homonymous hemianopia ought to have preceded the attack of total blindness, and ought again to have been observed for some time after the complete amaurosis had ceased. This objection, however, will be found to carry little weight, if we take into consideration that the individual concerned was of low mental capacity, that his sight had never been of the best, that the loss of sight was very sudden, and that his consternation at the time must have been very great indeed. These circumstances combined tend to prove that his power of observation was not very accurate.

The theory of the production in this case of complete amaurosis by a hemorrhagic focus in the left optic thalamus leaves several of the coëxistent symptoms without any explanation, but this is of little importance. The anosmia may have been of extra-cerebral origin. As to the ageusia, our knowledge of the conditions which give rise to it is so limited that we cannot be called upon to explain its origin in our case.

In conclusion we have to repeat that there is nothing in the record of the autopsy upon which could be based an explanation of the superior hemianopia observed during life. So much is certain that it was of extra-ocular origin. If we consider the interweaving of the optic-nerve bundles as described by Kellermann, and the fact that the uncrossed nerve bundles have been frequently observed to pass to the lower aspect of the optic nerves, it would seem doubtful, whether compression of the inferior portions of these nerves could produce superior hemianopia.





G. P. PUTNAM'S SONS, PRINTERS  
NEW YORK